115:—"The question now arises, do the cartilage and bone regenerate?" "They do not," he says emphatically. "After a period of from one to two years I have examined most of my old cases and have found the septum flaccid to the slightest touch of the probe. From one patient of fourteen, I removed a section and submitted to microscopic examination, and found only two muco-perichondria with a small amount of fibrous tissue between them." Dr. Freer, however, in his discussions of this same paper says he is not sure that such regeneration does not take place. He calls attention to certain cases seen by him some time after operation where he was fairly certain that there was greater firmness to the septum than would be given by the periosteum and perichondrum alone. However, there is so far as I know, no positive microscopical evidence on this subject. Personally, I have examined a number of such septa long after operation and though they were straight and functionating perfectly they certainly lacked underlying support of bone or cartilage. What is more, in the cases where I did replace a considerable part of the septum, the membrane was quite lax in other parts, showing that these replaced pieces had not proliferated, and completed the frame work.

It is a mooted question whether cartilage ever regenerates except from its perichondrium. To quote Ziegler, "Regenerative growth of cartilage after lesion either does not take place at all or in such insignificant proportions as to be without practical importance. On the other hand, new tissue is built up from the perichondrium."

Dr. M. Mutsucka, in Virchow's Archive for 1904, has an interesting article in which he has quoted the ablest opinion up to that time and there was difference in them as to the above question. Fedfer, Legros, and Gudden saw the cartilage regenerating by a division of the cartilage cells. Gussenbauer, Schwalbe, and Sieveking came to the conclusion that only the perichondrium took part in the regeneration. We would not then expect any material aid in the regenerative process from the regeneration in the replaced parts. The question has arisen as to whether on these bony or cartilagenous parts, replaced new tissue would be lain, thus leading to increase in the thickness of the septum. The surfaces lying next the periostium or perichondium are smooth and practically uninjured, and I do not believe there is new cartilage or bone deposited there. The results after nine months do not show any such condition.

REPORT OF CASES OF HEAD INJURY.*

By O. D. HAMLIN, M. D., Oakland.

In presenting this report of cases, the question of diagnosis and treatment will be mainly dealt with, in that the diagnosis and treatment of intracranial disturbances depends on a thorough and practical knowledge of the physiological functions and the anatomical locations of nerve centers.

Traumatic injuries of the head and diagnosis of same may cover symptoms of one or more conditions, such as concussion, laceration of brain tissue, compression of brain, extradural hemorrhage and subarachnoid serous exudate. Concussion and contusion of the brain associated with minute bruising of brain tissue will exist after all serious injuries of the skull.

The symptoms of concussion are varied according to the severity of the injury. Following slight concussion, the individual is stunned. There may be only a simple vertigo, or possibly mental confusion lasting but a short time. After a severe concussion there will follow a period of unconsciousness of a longer duration, the duration of unconsciousness depending upon the amount of injury to brain tissue. The sphincters may be relaxed and consequent involuntary micturition and defecation. The pulse will become slow and feeble along with the general systemic depression. The pupils still react to light. The temperature may be subnormal. It is impossible clinically to distinguish between concussion and contusion of the brain.

In serious laceration of brain tissue the symptoms of concussion may be present to a marked degree and will be characterized by immediate, pronounced and long-continued unconsciousness, often overlapping into unconsciousness produced by intracranial hemorrhage; moreover, in laceration of brain tissue, after recovery from the initial shock, fever may be present, which may rise to 103 or 104. Concussion alone is never associated with fever. Early fever is always the sign of laceration or lesions other than concussion. Mental irritability and restlessness will mark returning consciousness. Slight hemorrhages do not give us symptoms of compression, neither do slight depressions of cranial bones. In one case called to my attention, in which there existed hemorrhage from the middle meningeal and compression of the bony vault, no symptoms of compression were caused and patient was about for six days. Before symptoms of compression appear the cranial contents must be impinged upon to a very considerable extent. In extradural hemorrhage the most important symptom of traumatic intracranial hemorrhage is the interval of consciousness that exists from the time of the injury to the onset of unconsciousness. This period of consciousness may be preceded by the temporary or prolonged unconsciousness of concussion. In my opinion, in cases of hemorrhage which occur without an interval of consciousness, unconsciousness coming on immediately upon receipt of the injury, it must be that the injury is so severe that the unconsciousness caused by the concussion and laceration of the brain is continuous with the unconsciousness from hemorrhage. Therefore, with unconsciousness of long duration, when the temperature begins to rise, the pulse becomes slow and full, stertorous breathing sets in, the face is flushed and there is loss of reaction of the pupils and signs of paralysis of the upper motor centers or special senses, we must consider that the unconsciousness of concussion is continued over into the coma of compression. The sources of intracranial hemorrhage, whether from the middle meningeal or the middle cerebral, the veins of the pia mater, from the sinuses of the brain or from the lacerated brain tissue, can not be easily differentiated short of operative procedure. There is one condition not to be overlooked in connection with the question of hemorrhage, namely, the period of semi-consciousness,

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which sometimes follows concussion and laceration and gives rise to the suspicion of some more serious lesion.

For instance, J. G., aged 38, struck by one electric car while riding on the step of another, when seen at the hospital one-half hour later, was unconscious. There were no evidences of hemorrhage, no convulsive movements, no deviation of the eyes and no disturbances of the pupils; breathing was regular and normal and there were no disturbances in sensation or reflexes. Two hours later, the patient became partly conscious, knew his partner and recognized him sufficiently to tell where the key of the safe was. Twelve hours later and after a few hours' sleep, he became unconscious and symptoms of paralysis developed on his left side. In this case it may be seen that a period of nearly twelve hours' consciousness followed by unconsciousness might have been varied by a longer period of unconsciousness from concussion or the hemorrhage appearing earlier.

In fractures of the base of the skull, it is not uncommon to discover that what in the vault appears to be a simple fissure continues down to and involves the base of the skull. Fractures of the base of the skull are, however, regarded as more serious than those of the vault, because the cerebral disturbance is more pronounced, vital parts are endangered, and these fractures open into cavities which it is impossible to keep surgically clean. Danger of septic infection in such fractures is very great. According to McEwen, about 85% of basic fractures originate in the vault—that is, are caused by an extension of a linear fracture of the vault to the base.

In fracture of the base, hemorrhage may take place from the ear, from the mouth, from the nose or under the conjunctiva. Escape of cerebro-spinal fluid may be noticed from the nose or ear. In diagnosis of fractures of the skull, the signs usually present in fractures elsewhere are not available for diagnostic purposes. The hemorrhage is frequently so distinctly circumscribed as to mislead one to mistake the unresisting soft area which solidly defines margins for a depressed or even penetrating fracture. Hemorrhage from the ears may occur from other causes, such as injuries to the external auditory canal. When hemorrhage exists from the nose or ear, it may be accompanied by cerebro-spinal fluid and if collected, the presence of sugar may be demonstrated. It is also characterized by an extremely small amount of albumen and a relatively large amount of sodium chloride.

The differentiation between concussion and compression are fairly clear. In the case of concussion, the invasion is sudden, while in compression, it is usually slow. In concussion, the pulse may be slow and likewise feeble, while in compression, the lessened pulse rate is not marked by a corresponding diminution of force. In concussion, the pallor of the surface is marked, while in compression, the natural color is maintained. The respiratory act in concussion is not affected, while in compression the vagus center is often most decidedly affected. In concussion, the pupils respond to light though they may be unevenly contracted, while in compression, they are fixed, usually dilated and do not respond to light. Unconsciousness is common to both conditions, except in concussion unconsciousness is immediate, in compression later, unless one condition overlapses the other. Should symptoms of concussion persist, however, beyond those of the simple and temporary stun, artificial heat or administration of hot alcoholic drinks by the mouth, if the patient can swallow, otherwise by the rectum, inhalations of aqua ammonia or injection of 1/100 gr. of atropin to increase arterial pressure, or inhalation of nitrate of amyl to lessen the resistance of blood through the smaller vessels and capillaries. Under no circumstances should ice or cold water be applied to the head during this stage. As soon as re-action is established, all stimulating measures should be abandoned. With excessive re-action, the new line of treatment is indicated. Fullness of the cerebral vessels are indicated by a flushed face. Congestion of the conjunctiva and throbbing of the temples is to be met with the application of ice and cold compresses, at the same time the administration of active cathartics to lower blood pressure.

The treatment of compression of the brain depends upon its causes. The cause being removed, the brain usually recovers its functions, if its nutrition has not been disturbed. As a rule, ligation of small vessels causing hemorrhage of the brain is not necessary. Should it persist, however, the removal of a sufficient amount of bone to enable the vessel to be reached will be indicated and may be rapidly affected with Keen's gouge forceps, as the removal of bone even in the linear form causes very little callus during the process of repair. This is ascribed to the immobility of the fragments and subsequent very slight irritation present. This also explains the usual but not constant absence of symptoms of cerebral irritation, such as follow the presence of deposits of new bone in the inner surface of the cranial bones. Cases, however, occur in which disturbances of function result from the formation of bony deposits in this location and operative procedures are necessary for the relief of these. Complete regeneration following the loss of bone, either from accidental injury or from the use of the trephine, almost never occurs. The dura mater here assumes the function of a periosteum to a minor extent, as shown by the fact that excessive formation of callus, under these circumstances, is almost unknown.

The question of how to lower blood pressure during operations on the head has been a very broad one and has been very widely discussed. Bleeding has been suggested, sitting the patient in an upright position has been suggested, and recently Dr. Robert H. M. Dawbarn of New York has written an article entitled "Sequestration Anæmia in Brain and Skull Surgery."[1] His method is as follows: Tourni-

1. In the February, 1907, number of "Annals of Surgery."

quets are placed around each thigh and arm close to the trunk, a towel being placed under the tourniquet to cause pressure of a wider area. It must nearly stop the venous but not the arterial current. In this way the patient is bled into his own limbs. Dr. Dawbarn, in his paper, states the danger of prolonging the use of this tourniquet during this operation.

Case I. W. C., injured August 18, 1906. Seen at hospital at 1:45 p. m. Temperature 99.4. Pulse 80. Injured about 8 p. m. the day previous, August 17th. Contusion and laceration of scalp above right ear about two inches in diameter. Patient had been up and around during the day of August 18th, but was rendered unconscious for about two hours after injury on August 17th, which occurred from being hit by switch engine at railroad yards. Pupils equal and reacted to light. Respiration normal. Heart action regular. Pulse full. Examination of injury showed slight laceration of scalp over right ear and great tenderness on pressure and a large bulging mass about two inches in diameter containing fluid. Horseshoe-shaped incision of scalp revealed a large hematoma and several loose fragments of temporal bone. On removal of depressed fragments a very profuse hemorrhage followed. Loose fragments were quickly cleared away and a large extradural clot found. On examination of dura no normal pulsation could be seen. Dura was opened and considerable effusion relieved, after which normal pulsation returned. Sharp edges of bone were carefully removed and plain gauze packing used as dressing. Following day, temperature 100; pulse 80. Patient dressed every day. One drainage strip was not removed until four days later, and then partially replaced to give additional drainage if necessary. Patient discharged from hospital September 1, 1906.

Case II. E. C., Spaniard, aged 42. Examined at hospital November 13, 1906. Was struck by street car while crossing track three days before. History showed that patient remained unconscious two and one-half hours, later returned home and was up and about, did not feel well, complained of dizzy spells and pain in left side of head when seen at hospital. Examination of head showed tenderness and bulging in left frontal region. Pulse 120. Patient was prepared for exploratory incision. Horseshoe-shaped incision was made over left frontal region. A large hematoma of scalp and linear fracture running through left frontal bone was found. Small trephine was used in line of fracture and opening enlarged by use of Keen's gouge and de Vilbus used along line of fracture. A large extradural clot was removed from under line of fracture about one and one-half inches in diameter and about one-half inch thick. Dura was exposed and did not pulsate. Dura opened and considerable serus exudate relieved. Clot and exudate were removed, wound was dressed with plain gauze and drained. Patient lifted in bed to an angle of forty-five degrees for the purpose of lowering blood pressure. Two days later the patient's temperature was 99, pulse 80 and condition generally good. Three days later temperature was normal, pulse 70. Three weeks later, patient was discharged.

Some points in this case to which I desire to call attention are the use of the de Vilbus forceps, the presence of a cerebral exudate under clots, and the position of the patient in the after-treatment.

It is easy, by running the small de Vilbus blade along a linear fracture, to rapidly open the skull for exploratory purpose and reveal a clot or point of maximal hemorrhage, and determine quickly the necessity for or point of application of the trephine. This procedure may be carried out over any part of the brain surface without injuring sinuses or brain tissue.

When, after an extradural clot has been removed, the pulsation of the dura does not return, a sub-dural cerebral exudate is usually present and an opening to remove this exudate has not, in my experience, been followed by any serious results, unless laceration of brain tissue existed and bulged through opening in the dura. If there is danger of this complication, the patient should be carefully watched and on occurrence of a hernia cerebri appropriate treatment should be adopted.

In the treatment of injuries of the head, position is a very important factor. In concussion or other condition in which cerebral anæmia exists, the head must be low, but otherwise the head must be raised after the effects of the anesthetic have passed off, in order to lessen the tendency to hemorrhage and exosmosis due to engorgement of the cerebral vessels.

Case III. F. M., injured May 11, 1906. Inventor, aged 23 years. Fell from a buggy. When seen one hour later at hospital, fracture of lower jaw on both sides at angles. Patient unconscious. Pupils reacted to light and were equal. No manifestation of motor disturbance. Patient restless. Temperature 95; pulse 40. No evidence of cerebral hemorrhage. Stimulation treatment instituted. No attempt made to wire or reset jaws, as patient was not in condition to stand an anaesthetic. Temporary dental splints and chin bandage with large hole in center of dental splint for feeding patient used with expectation of adjusting permanent splint after patient's condition improved. At midnight on May 11th patient's condition still remained the same. Patient would take nourishment. Bowels well emptied by enema and urine drawn by catheter. Next day, the 12th, no change in physical or mental condition; temperature 96, pulse 52. At 11 o'clock, on May 12th, temperature 99, pulse 62. Patient more comatose. The next day, May 13th, at 11 o'clock, patient decidedly comatose; temperature 101, pulse 68. Patient generally in a dying condition. Patient died at 3 p. m. Temperature, just before death, 102. This patient's temperature at this time was probably due to compound comminuted fracture of the jaw, with possible infection or absorption, as tissues about fracture were severely contused and lacerated.

Autopsy showed no fracture of skull and no cerebral hemorrhage, but there was extensive generalized congestion of brain tissue, and excessive exudate into the ventricles, especially the fourth.

Case IV. M. A., Italian, November 22, 1906. Residence, Niles. Brought to me by Dr. Morrison. Examination showed equal pupils, which reacted to light. Temperature 102. Pulse 126. Respiration 38. Paralysis of right side, right arm and leg and speech. Struck, night of November 21st, about 10 p. m., on right side of head, with bottle. History shows that patient returned home three miles from Niles in his own buggy, driven by himself, unhitched his horse, put his wagon in the shed and entered his house (it was then about 11:45 p. m.), sat at the table with his wife, told her of a row with a certain man and suddenly stopped speaking. Dr. Morrison was sent for and gave him treatment during that night. Next afternoon Dr. Morrison brought the patient to Oakland and placed him under my care. Exploratory incision was made over point of injury on right side, but no depressed fracture found. Incision was carried over to left side of head, where fracture extended across the top of the vault and over the fissure of Rolando. The opening was made through

the bone over the fissure of Rolando and a large clot in the fissure exposed. The reason for the right-sided paralysis was thus evident. Clot was removed and immediately on recovering from the anaesthetic patient's leg was relieved of its paralysis. Hand and voice still remained paralyzed. Later speech greatly improved, but condition of hand was unchanged. Brain tissue being greatly disorganized from pressure of clot, of which three and a half to four ounces were removed, patient lived several weeks, but gradually became weaker and died.

Autopsy showed disorganization of brain tissue and general cerebritis.

Case V. Mrs. G. B., aged 30 years, housewife. Injured in street car accident. Seen two hours after accident in hospital. Examination revealed severe abrasion over left side of face and swelling and abrasion over left side of forehead and fracture of the acromial process of left shoulder. Patient partly unconscious. Pupils equal; react to light. Rectal temperature 96. Pulse 94. Incision across scalp revealed a linear fracture running anterior posteriorly through left side of frontal bone. Fracture seemingly very slight, no displacement of bones, no evidence of hemorrhage. Scalp wound sewed up and patient put to bed and observed closely for further symptoms. Next day, September 27th, patient very restless; partly conscious. Pupils equal; react well to light. No evidence of motor disturbance. September 28th, patient still very restless and partly conscious. Temperature 100 2-5. Pulse 100. Patient took liquid nourishment. September 28th, patient's condition about the same, but more evidence of consciousness. Pulse stronger. Recognized members of family. Patient's mental condition gradually but slowly improved from this time. Slight purgation and ice cap at this time, as the flushed condition of the patient's face, together with character of pulse, showed evidence of rise of cerebral blood pressure. At no time was there evidence of motor disturbance. The slow but gradual improvement in the mental condition kept up, and the patient left hospital November 4, 1906, with mental condition good, but very nervous and weak.

The interesting point in this case was the difficulty that arose in the diagnosis of the fracture of the acromial process in a partially unconscious patient. An injury of the right motor area by contracoup was at first suspected, on account of the disability of the left arm, but further careful examination showed that this was due to an injury of the acromial process.

In an unconscious patient and where the details of the accident are unknown, disturbance in the functions of limbs are very important aids in localizing the point of maximal cranial and cerebral injury, but it must be definitely decided by very careful examination that such a motor disturbance is not due to local rather than central injury. Sometimes, when there is no abrasion or discoloration of the skin, fractures about the hips, shoulder or other parts can be readily overlooked.

Case VI. Mrs. B., American, aged 40 years, housewife. Found unconscious on the street, October 4, 1906. Had been riding in buggy. Severe contusion of the right side of the back of head at base of skull. Patient unconscious. Pupils unequal, right pupil dilated. left pupil not so much so. Both pupils react to light. Temperature 98; pulse 60. Cheyne stokes respiration. Hemorrhage from right ear. Examination revealed no motor disturbance. Patient prepared for exploratory incision. Cavities of nose and ears thoroughly cleansed, right ear irrigated with very hot boracic acid solution and packed with iodoform gauze. Incision made over point of injury and compound comminuted fracture of base below lateral sinus on right side about one and a half inches in diameter exposed. Fragments of bone removed and laceration of dura and subdural hemorrhage found. Sinus apparently not injured. Bony fragments and blood clot removed. Patient put to bed, given 1/50 gr. of strychnin every four hours, 10 min. adrenalin and 1/150 gr. atropin, hypodermically, and observed for further symptoms. October 5th, temperature 97, pulse 100, respiration 32. Enema given of coffee, brandy and saline every four hours. Patient still unconscious. Dressings of head showed evidence of moisture. Head redressed; large quantity of blood of watery character. October 6th, patient's temperature 100 3/5, pulse 102, respiration 30. Patient still unconscious. Passed urine and feces in bed, notwithstanding bladder being emptied by catheter every six to eight hours. No longer retained rectal nourishment. Patient had three convulsions lasting from five to ten seconds. Head redressed and a large quantity of blood again removed. October 7th, temperature 100 2/5, pulse 100, respiration 34. Six convulsions during night, lasting from one to two minutes. Head redressed; less blood. Patient partly conscious. Patient makes effort to talk, and did answer "Yes" and "No" in a whisper. October 8th, temperature 102, pulse 120, respiration 40. Patient not so conscious as the day before. Holds nutritive enema at times. Up to this time, patient was given nothing by mouth, as it was impossible to get her to swallow. Head redressed; very much less seepage. Patient's face more flushed and showing more evidence of cerebral congestion. Patient raised in bed to an angle of forty-five degrees to lower blood pressure and ice cap applied. From October 8th to October 12th condition remained about the same. Still unable to take nourishment by mouth. Eight to ten convulsions in twenty-four hours, but occurring further apart and of less severity. October 13th, patient begins slowly to take small quantities of liquid food by mouth. No convulsions. Answers questions in a very low whisper. From October 13th to 20th temperature ranges from 98 to 100. Still troubled with involuntary evacuations at times, probably once or twice in twenty-four hours. Patient's general condition improved and was discharged from hospital November 9, 1906. Last seen February 1st; apparently well.

Case VII. J. M., American, aged 27 years, clerk. Seen at hospital January 21, 1907. Injured January 20th, about 11 p. m., being hit on the head by the controller of electric car. Patient up and about all day of 21st, suddenly became unconscious at his home about 7 p. m. Removed to hospital. On examination patient showed no disturbance of motor areas. Pupils equal, respiration regular, temperature 98, pulse 100. An exploratory incision made over injury on right side of frontal region. Found depressed fracture. Removed fragments of bone and extradural clot. On opening dura considerable serus exudate. Head dressed, patient put to bed, but elevated to an angle of forty-five degrees, applied ice cap. Head dressed every day. Patient given liquid diet. Left hospital two weeks later.

Case VIII. B. L., Italian laborer, aged 42 years. Struck by runaway team, December 22, 1906, at 7:30 p. m. Examination showed fracture of occipital bone and several extensive lacerated scalp wounds in occipital region. Patient unconscious. Hemorrhage from both ears. Pupils dilated; did not react to light. Pulse 60, temperature 97. Scalp wounds were enlarged and injury to bone examined. Extensive hemorrhage about cerebellum. Loose pieces of bone, which were about two inches in diameter, removed and clot cleared away and hemorrhage apparently stopped. December 23rd, patient in a comatose condition. Pulse 126. Temperature 100. Pupils still dilated; no reaction to light. Patient delirious.

General condition much worse. Patient died at 11:30 a. m.

Autopsy showed laceration of cerebellum and extensive hemorrhage about the base of brain and in the fourth ventricle.

Case IX. K. C., aged 12 years. October 10, 1906. Injured by being struck by automobile. Temperature 98 1/5, pulse 75. Pupils equal; react to light. Extensive laceration of right cheek. Scalp wound over right parietal region three inches in length and large hematoma over median line of scalp. Child partly unconscious. Examination showed no motor disturbance. Exploratory incision made; large hematoma found under scalp in median line. Having no reason for further interference, scalp was sewed up and patient put to bed. Next day, temperature 98 2/5. Patient continued to improve and was discharged from hospital in ten days. No need for operation in such case other than exploratory incision to determine what was under hematoma.

Case X. M. L., aged 14 years. Struck by a runaway team. Extensive laceration of scalp over right parietal region. Temperature 97, pulse 128. Pupils equal. No motor disturbance. Incision revealed depressed bone 1¼ inch in diameter in right parietal region about over fissure of Rolando and extradural clot. Loose fragments of bone and clot removed. Brain pulsating. No further symptoms developed.

Case XI. F. S., aged 41 years. November 28, 1906. Injured by falling from an automobile. Examination showed pupils equal; reacted to light. Hemorrhage from right ear. Temperature 97, pulse 54, respiration 22. Patient unconscious. No laceration of scalp, but slight contusion over median line on top of head about two inches in diameter. Accident happened about two hours before. Examination showed no evidence of motor disturbance. Exploratory incision over parietal region revealed no fracture of the skull. History showed patient to be very much intoxicated at time of injury and had been so for several days. Patient put to bed to await further developments. Following day, November 29th, pupils equal; react to light. Temperature 99 3/5, pulse 125, respiration 30. Patient very restless; attempts to talk. Still can not recognize any one. General relaxation of limbs. Reflexes all diminished. November 30th, patient comatose. Pulse 130. Temperature 100 2/5. Patient generally in a dying condition. Patient died at 12:30 p. m.

Autopsy showed fracture of the right outer border of the forearm magnum at the condylar portion where it articulates with the articular process of the atlas and fracture into the anterior condylar forearm which transmits the hypoglossal nerve and a branch of the ascending pharingeal artery, the posterior condylar forearm which transmits a branch from the lateral sinus being uninjured, and very extensive hemorrhage in the cord about the medulla. This hemorrhage extended down into the spinal cord subdurally. Dr. Harvey Cushing and Dr. M. Allen Starr of New York, in the A. M. A. Journal of September 22, 1906, have suggested lumbar puncture in the diagnosis of cerebral hemorrhage of asphyxiation, of new-born infants from delayed labor. The lumbar puncture shows a bloody cerebral spinal fluid in such cases, and I think the lumbar puncture may be of equally great importance in diagnosing fractures of the base. I have used lumbar puncture and find it a good means of diagnosis of hemorrhage of base, as spinal fluid shows pressure of blood.

Case XII. J. R., aged 30 years. Injured September 26, 1906, by falling from a horse. Seen five days later, October 2nd, at hospital. Patient had been up and around until day before, October 1st. Complained of headache. Scalp wound had been dressed and sewed up by a physician who had seen him at time of injury. Had not had any treatment since. Patient showed no signs of motor disturbance, but complained of headache. Patient remained in bed on day of October 2nd, and about 8 a. m. had a convulsion. One-half hour later had another convulsion. Was called to see patient, and during examination of wound, which was opened and stitches removed, patient had convulsion, which appeared to be Jacksonian epilepsy, wound being in left frontal region. Patient was prepared for exploratory incision. The incision was made over frontal region, which revealed a depressed bone about 1¼ inch in diameter over left frontal region and extradural clot. After removal of fragments of bone and extradural clot, brain showed no evidence of pulsating. Small incision was made into dura and considerable exudate, which seemed to be walled off about injury, removed. On removal of fluid brain showed normal pulsation. Patient showed no further trouble. Wound dressed every day. Temperature never rose above 100 and pulse rated from 90 to 110. In two weeks was discharged from hospital, convalescent.

Case XIII. Jas. H., aged 19 years, carpenter. Injured March 2, 1907. While running upstairs, hit head against joist, fell over side of stairs about ten feet and remained unconscious for a short time. Was taken home in about one hour and Dr. Koford called. Later I was called in consultation with Dr. Koford, and after head being shaved a small laceration one-quarter inch in length was found on right parietal bone about right parietal eminence. Careful examination of wound and probing showed no evidence of fracture about point of laceration, but two inches to the left of small laceration and distinct crepitus for about two inches. Case was diagnosed as a fractured skull and prepared for an exploratory incision. Exploratory incision revealed a crushed leadpencil under the scalp and no fracture of the skull.

Case XIV. N. T., aged 75 years; news agent; birthplace unknown; resident of California for forty years. Injured on March 31, 1907, at 10 a. m.

Injured by electric car; was knocked down and struck head on ground. Examination showed only slight contusion over left cheekbone. Patient unconscious; remained in deep coma until death; Pupils did not react to light; pulse about normal; labored breathing; complete relaxation, with no reaction to stimulants. Patient lived about five hours.

Autopsy showed anemia of the brain, due to concussion; also a slight hemorrhage into right cerebral fossa over temporal lobe, but did not enter into the cause of death. Death due to concussion and shock.

Discussion.

Dr. J. H. Barbat, San Francisco: The localization of brain injuries is comparatively easy when they are located in the motor area, but it is quite different when the anterior portion of the frontal lobes is affected. We may have extensive laceration of the brain tissue in this region without the slightest positive symptoms. I saw a young man who fell 20 feet and landed on his hands, breaking both wrists, recover from the fractures and go home four weeks from the date of injury. Three days later, the head symptoms developed and he died inside of five weeks from the fall, with symptoms of meningitis. The autopsy showed a fracture of the base of the skull from the horizontal plate of the frontal bone to the foramen magnum. The chibriform plate of the ethmoid was crushed in and the ethmoid cells opened through the frontal bone. The ethmoid cells contained a piece of brain tissue as large as the end of the thumb; no hemorrhage or symptoms of meningities. In this case more than four weeks elapsed without the slightest symptoms pointing to the injury of the brain.